

Right ventricular dysfunction as a predictor of mortality in patients with sepsis or septic shock in the emergency department

Right ventricle and mortality in sepsis

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Abstract

Aim: Sepsis and septic shock present a significant challenge in the emergency department (ED) owing to their high mortality rates. The presence of right ventricle (RV) dysfunction during sepsis could potentially exacerbate unfavorable outcomes. However, the precise relationship between RV dysfunction and mortality in septic patients remains poorly understood. Therefore, this study aims to investigate the prognostic value of RV dysfunction as a predictor of mortality in patients presenting with sepsis or septic shock in the ED of Alexandria main university hospital.

Material and Methods: A prospective observational study was performed on 75 patients who presented to the ED with sepsis or septic shock. Echocardiographic parameters, including the tricuspid annulus systolic plane excursion (TAPSE) and the fractional area change, were measured within 24 hours after admission for assessment of RV dysfunction; then, patients were evaluated according to sequential organ failure score (SOFA) and acute physiologic and chronic health assessment (APACHE). Using multivariate regression, the current study assessed the relationship between 28-day mortality and the presence of RV dysfunction, SOFA, and APACHE.

Results: There were 75 patients in the study. 22 patients (29%) had RV dysfunction. When compared to patients without RV dysfunction, those with RV dysfunction had a greater mortality rate (68% vs. 34%, $p<0.001$). RV dysfunction was shown to be associated with mortality in univariate regression analysis ($OR:4.16$, $CI:1.44-12$, $P<0.008$), and multivariable regression analysis revealed a high correlation between RV dysfunction and mortality ($OR:3.98$, $CI:1.3-11.9$, $P<0.013$). With a statistically significant area under the curve, sensitivity, specificity, positive predictive value, and negative predictive value of 0.64, 45.5, 83.8, 68.2, and 66%, respectively, the receiver operator curve (ROC) indicated that RV dysfunction was linked to a considerable mortality rate.

Discussion: RV dysfunction was found in nearly one-third of the studied patients with sepsis and is associated with fourfold higher 28-day mortality.

Keywords

Right Ventricular Dysfunction, Sepsis, Septic Shock, Sepsis-Induced Cardiomyopathy, Mortality, Echocardiography

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Introduction

Sepsis is defined as “life-threatening organ dysfunction caused by a dysregulated host response to infection” [1]. The emergence of cardiac failure is one of the most significant events that occur during sepsis, as it directly relates to morbidity and mortality, it is also known as Sepsis-induced cardiomyopathy (SICM) [2]. SICM is common and poses a significant load on the healthcare system where studies show that 40% to 50% of sepsis patients have myocardial damage [3].

There is no single definition that is universally accepted for SICM, despite the fact that many studies have shown evidence of circulatory dysfunction in septic patients [4]. However, the primary feature of SICM is contractile dysfunction [5]. It presents with: dilatation of both ventricles, Left ventricular (LV) systolic and/or diastolic dysfunction, and/or right ventricular (RV) impairment [6].

The majority of studies have been almost exclusively concerned with LV function. On the other hand, the RV is neglected in research, given its smaller muscle mass and single organ function. Nevertheless, the RV is a vital part in circulation as it is the second-largest pump in the circulatory system and is connected in series with the LV. Additionally, RV plays an important role in SICM.

During sepsis, the RV suffers from diminished filling, reduced contractility, and increased afterload. This will lead to a failure of the RV output to match the increased LV afterload as a result, CO will decline due to decreased LV preload [7]. Ventricular interdependence results from the RV and the LV being connected not only in series but also by a common septum [8].

Transthoracic echocardiography (TTE) is a minimally invasive, bedside method that may provide repeatable estimations of RV size and contractility [9]. Compared to the LV, imaging the RV is more challenging. As a result, there continues to be a divergence of opinions regarding the selection of echocardiographic parameters to define RV dysfunction.

The results of recent studies evaluating RV dysfunction were variable; whereas some showed no difference between survivors and non-survivors [10], others discovered a significant incidence of RV dysfunction that was linked to short and long-term mortality [11, 12].

Due to the fluctuating nature of these findings, there remains a necessity for a more dependable characterization of RV dysfunction in sepsis and septic shock, applicable for both research and clinical purposes.

The hypothesis of this study posits that the presence and severity of RV dysfunction upon presentation to the ED will be positively correlated with mortality in patients with sepsis or septic shock.

Material and Methods

This prospective observational study was performed on a registry of 75 patients who presented to the ED of Alexandria main university hospital (AMUH) with sepsis or septic shock. The aim of this study was to investigate the prognostic value of RV dysfunction as a predictor of mortality in patients presenting with sepsis or septic shock in the ED of AMUH.

Sample Size

Using the Power Analysis and Sample Size (PASS 2020) software (NCSS, LIC, Kaysville, Utah, USA, ness.com/software/pass), the Department of Medical Statistics, Medical Research Institute, Alexandria University, calculated the sample size while accounting for the 5% level of significance and the 5% precision using the Z-test [13].

Inclusion criteria

Adult patients over the age of eighteen and admitted to the emergency department (ED) with sepsis or septic shock, using the operational definitions provided by the surviving sepsis campaign (2021) [1].

Exclusion criteria

Poor echo window, moderate or severe mitral and/or aortic

Table 1. Comparison between survivors and non survivors according to different ECHO parameters

ECHO	Total (n = 75)	Survivors (n = 42)	Non survivors (n = 33)	Test of Sig.	p			
TAPSE	19.0 (16.0 – 23.0)	23.0 (19.0 – 24.0)	18.0 (14.0 – 19.0)	U=223.50*	<0.001*			
FAC	42.36 ± 9.12	44.50 ± 8.31	39.64 ± 9.50	t=2.362*	0.021*			
RV ED area	19.41 ± 4.47	20.16 ± 4.56	18.45 ± 4.24	t=1.664	0.100			
RV ES area	10.70(8.85 – 12.75)	10.70(8.7 – 12.8)	10.30(9.2 – 11.8)	U=650.0	0.646			
EF (%)	55.72 ± 14.29	54.14 ± 15.07	57.73 ± 13.19	t=1.097	0.276			
E	93.87±18.57	91.30±18.89	97.15±17.91	t= 1.361	0.178			
A	67.70±8.75	67.16±9.19	68.38±8.24	t= 0.597	0.552			
E/A ratio	1.36±0.24	1.32±0.27	1.42±0.17	t= 1.7495	0.077			
Lateral E'	7.40 (6.2-9.1)	7.40 (6.5-7.9)	6.70 (4.9-11.3)	U= 667.5	0.785			
E/E'	12.10 (8.6-17.3)	12.05 (10.2-13.5)	16.82 (7.2-21.8)	U= 672.0	0.823			
RV dysfunction	22	29.3%	7	16.7%	15	45.5%	X ² =7.388*	0.007*
LV systolic dysfunction	27	36.0%	16	38.1%	11	33.3%	X ² =0.182	0.670
LV diastolic dysfunction	37	49.3%	20	47.6%	17	51.5%	X ² =0.112	0.738
LV dysfunction	55	73.3%	31	73.8%	24	72.7%	X ² =0.011	0.916
Combined LV&RV dysfunction	10	13.3%	3	7.1%	7	21.2%	X ² =3.166	0.075

Qualitative parameters were expressed as number and percent, while normal distributed quantitative parameters were expressed as median (interquartile range)

t: Student t-test

U: Mann Whitney test

c2: Chi square test

p: p value for comparing between Survived and Died

*: Statistically significant at p ≤ 0.05

valve disease, regional wall motion abnormalities suggesting regional ischemia or prior infarction, pulmonary hypertension or cor-pulmonale, and pregnant females.

Informed Consent was obtained from each patient participating in the study or from their family, after providing detailed explanations about the research's objectives and procedures.

Throughout the study, strict confidentiality measures were upheld to safeguard the privacy of all individuals involved.

Complete history taking, a thorough physical examination upon admission that included the Glasgow Coma Scale (GCS) [14], vital signs, oxygen saturation, central venous pressure, and urine output were all performed for each patient. Upon admission, an electrocardiogram (ECG) and arterial blood gases were done, and the hypoxemia index ($\text{PaO}_2/\text{FiO}_2$) was calculated.

On the first day of hospitalization, scores for the quick sequential organ failure assessment (qSOFA) [15], the sequential organ failure assessment (SOFA), and the acute physiologic and chronic health evaluation (APACHE II) scores were calculated [16].

The criteria for the Surviving Sepsis Campaign in 2021 were followed during the treatment of the patients [1].

All hemodynamic, respiratory/ventilatory, vasoactive therapy,

Table 2. Multivariate logistic regression analysis for the parameters affecting mortality (n = 33 vs. 42) model 1

	p	OR (LL - UL 95% C.I)
APACHE II	<0.001*	1.282(1.152-1.426)
SOFA	<0.001*	1.547(1.265-1.891)
Presence of RV dysfunction	0.013*	3.988(1.332-11.940)
Presence of LV systolic dysfunction	0.187	1.954(0.723-5.280)
Septic shock	0.001*	11.161(2.761-45.117)
lactate	<0.001*	2.183(1.455-3.277)

OR: Odd's ratio C: Categorical variables

C.I: Confidence interval

LL: Lower limit UL: Upper Limit

*: Statistically significant at $p \leq 0.05$

Adjusted by age and sex

and fluid balance were recorded.

Using a 2.8–4 MHz phased array probe and a Vivid e machine (General Electric, Boston, USA), all patients were investigated by TTE performed by an emergency physician during the first 24 hours of diagnosis.

RV assessment

RV fractional area change (FAC) and tricuspid annular plane systolic excursion (TAPSE), assessed by M-mode, are used to evaluate the RV function from the apical four-chamber 2D perspective. RV dysfunction is characterized by a TAPSE of less than 1.6 cm or a FAC of less than 35% [17].

LV assessment

In the apical four and two chamber view, the modified biplane Simpson equation was used to calculate the left ventricular ejection fraction (LVEF). From the apical four-chamber view, the peak mitral inflow E and A velocity waves on the pulsed wave Doppler, the E/A ratio, and the E wave deceleration time (EDT) were measured. Using pulsed wave tissue Doppler imaging at the lateral mitral annulus on the four-chamber apical view, the diastolic e' peak velocity was determined, and E/e' was computed. LV systolic dysfunction was defined as $\text{LVEF} < 45\%$. $E/e' < 13$ was the definition of LV diastolic dysfunction[18]. Systolic or diastolic dysfunction is referred to as LV dysfunction [19].

Statistical analysis of the data

The computer was given data, and IBM SPSS software package version 20.0 was used for analysis. (IBM Corp., Armonk, NY) Numbers and percentages were used to describe the qualitative data. To confirm that the distribution was normal, the Shapiro-Wilk test was performed. The terms range (minimum and maximum), mean, standard deviation, median, and interquartile range (IQR) were used to characterize quantitative data. At the 5% level, the results' significance was assessed.

Ethical approval

This study was approved by the Ethics Committee of the Alexandria University (Date: 2021-10-20, No: 0201570).

Results

128 patients were assessed in this study, 53 patients were excluded while 75 patients were enrolled; 23 patients had sepsis (31%), whereas 52 patients had septic shock (69%), 75% of whom were mechanically ventilated, and 64% of whom received vasopressors. Overall mortality in the study was 44%. The mean length of the ICU stay was 10 days.

A statistically significant difference between survivors ($44 \pm 8\%$) and non-survivors ($40 \pm 9\%$) regarding FAC was observed, with a mean value of $42 \pm 9\%$. In addition, there was a statistically significant difference between survivors (21.2 ± 3.9 mm) and non-survivors (15.7 ± 4.5 mm) regarding TAPSE, with a mean of 18.8 ± 5 mm for all cases. (Table 1).

RV dysfunction was present in 22 patients (29%). Similarly, LV systolic dysfunction was found in 27 patients (36%), and LV diastolic dysfunction was found in 37 patients (49.3%). LV dysfunction (systolic & diastolic) was found in 55 patients (77%). Only nine patients (19%) had isolated RV dysfunction, despite the significant overlap we found between LV and RV dysfunction (Table 1).

Patients with RV failure had lower ventilator-free days (2 vs.

Figure 1. ROC curve for APACHE II, SOFA, TAPSE and FAC to predict mortality (n = 33 vs. 42)

7.8, $p < 0.001$) and increased mortality compared to patients without RV dysfunction (68% vs. 34%, $p < 0.001$). Mortality and RV dysfunction were correlated (OR: 4.16, CI: 1.44-12, $P < 0.008$), but not with LV diastolic dysfunction (OR: 1.16, CI: 0.46-2.9, $P < 0.73$) or LV systolic dysfunction (OR: 0.5, CI: 0.18-1.3, $P < 0.16$), according to univariate regression analysis. RV dysfunction and mortality had a high correlation according to the multivariate regression analysis (OR: 3.98, CI: 1.3-11.9, $P < 0.013$) (Table 2). Regarding the dysfunction of the RV, where an area under the curve, sensitivity, specificity, positive predictive value, and negative predictive value were 0.64, 45.5, 83.8, 68.2, and 66%, respectively, that was statistically significant, the receiver operator curve (ROC) indicated that it was related to substantial mortality. A similar outcome was obtained with TAPSE and FAC, with an APACHE II score of > 18 and an initial sofa score of > 9 (Figure 1).

Discussion

The study enrolled 75 patients who were admitted to the ICU with sepsis or septic shock. To assess RV dysfunction, different cut-offs derived from echocardiography and pulmonary artery catheterization were used in previous studies. In the current study, thresholds were chosen following published guidelines where, TAPSE < 16 mm or FAC $< 35\%$ were used to define RV dysfunction [17]. Those parameters were selected because they are widely used in clinical practice and are simple to measure. The same definition was used by Lanspa et al.[19], a similar definition was used by Innocenti et al., [20] who defined RV dysfunction as TAPSE < 16 mm.

The current study involved 22 patients; 29.3% of patients were suffering from RV dysfunction, either isolated or combined with LV dysfunction. Other studies have agreed with the current study that RV dysfunction is common in sepsis, Innocenti et al. [20], found RV dysfunction in 34% of patients, and Kim et al., [21] reported that 40.7 % of patients had RV dysfunction.

The risk of mortality was four times greater in patients with RV dysfunction. It was discovered that RV dysfunction contributed independently to mortality. Our findings support and add to existing recent research on RV in sepsis. In a meta-analysis of observational studies, Vallabhajosyula et al.[22], discovered a correlation between RV dysfunction and higher mortality over the long term (OR 2.26, 95% CI 1.29-3.95, $P = 0.004$) as well as the short term (OR 2.42, 95% CI 1.52-3.85, $P = 0.002$).

This correlation can be explained by the increased cardiomyocyte cellular dysfunction within the dysregulated humoral metabolic context of sepsis, which is thought to be the pathophysiology of the observed RV dysfunction in septic patients in the present study and others. However, some other postulated mechanisms deserve discussion.

First, RV dysfunction may be an indirect measure of the severity of lung disease, which is the direct cause of death in those patients, since RV is impacted by the afterload exerted by the pulmonary circulation.

Second, the administration of catecholamines may have directly contributed to the mortality by causing RV dysfunction; in other words, RV dysfunction may have been a bystander to the increased mortality rather than the cause.

Consequently, rather than being a result of an underlying cardiac issue, RV dysfunction in sepsis may simply be a side

effect of the overuse of vasopressors and fluid administration during the sepsis process.

In the current study, there was no significant difference between patients with RV dysfunction and patients without RV dysfunction regarding the need for mechanical ventilation (MV), PaO₂/ FiO₂, or the need for vasopressors. There was a significant difference regarding the vasopressor dose [5].

The current observational research did not standardize the time of fluid delivery, or TTE. Nevertheless, we were unable to find any meaningful difference in the cumulative balance between individuals who had RV dysfunction and those who did not. This demonstrates that different methods of administering fluids are not the main reason behind RV dysfunction.

Previous research has demonstrated that RV dysfunction has a separate effect on short-term mortality. In patients with and without RV dysfunction, the rates of acute respiratory distress syndrome (ARDS) were similar in the meta-analysis conducted by Vallabhajosyula et al.[22], although the rates of invasive MV were lower.

According to a different study by Vallabhajosyula et al. [23] patients with and without PHTN experienced similar levels of illness severity, including the need for MVs and ARDS.

Although the current study revealed a strong correlation between high short-term mortality and RV dysfunction, whether it occurred alone or in conjunction with LV systolic or diastolic dysfunction, mortality was not linked to LV systolic or diastolic dysfunction by itself.

In line with this finding, Lanspa et al.[19] discovered no correlation between mortality and LV diastolic or systolic dysfunction. They stated that both LV diastolic dysfunction (OR 0.94, CI 0.54-1.61, $P = 0.81$) and LV systolic dysfunction (OR 0.92, CI 0.57-1.5, $P = 0.73$) did not correlate with death, according to their univariate regression analysis.

Additionally, Kim et al.[21], produced similar findings, concluding that LV dysfunction (systolic and diastolic) was prevalent. Nevertheless, the only condition linked to mortality was RV dysfunction.

The inconsistent connection between RV and LV (either systolic or diastolic) and mortality in this study raises concerns because previous research has shown increased mortality.

The criteria of EF $< 45\%$ employed to identify LV dysfunction are probably very sensitive and may detect even minor cases of LV dysfunction, despite adhering to established standards; employing a lower threshold might have yielded different associations.

On the other hand, the thresholds for RV dysfunction (FAC $< 35\%$ and TAPSE < 16 mm) may exclusively flag severe cases of illness and could potentially target a subset of patients who are more critically ill.

Furthermore, even though LV diastolic dysfunction has been linked to mortality in septic patients, a correlation in this group of patients was not found. This could be because of the study's strong signal for RV dysfunction, the low prevalence of isolated diastolic dysfunction, or the link between diastolic parameters and fluid intake. Lastly, the TTE, or fluid reception time, of our study may be different from that of other studies.

Study strength

This study focused on the RV's echo parameters, and the association between RV dysfunction and mortality. The scarcity

of research focusing on the right ventricle makes it a point of strength in this study.

Limitation

Several limitations have been encountered during the process of conducting our study. Since this is a single-center study, patient selection and treatment may have been impacted by regional management practices. The study was limited by its observational nature, which hinders any causal claim about the link between RV dysfunction and survival in sepsis patients.

We did not have enough pre-sepsis information on our patients to evaluate whether the RV dysfunction was caused by sepsis or if the patients had underlying RV dysfunction, even when individuals with clear chronic heart disease were eliminated.

Patients with RV dysfunction in this research tended to be older, to have more severe illnesses, and to have more concomitant conditions. These variables could distort the correlation between RV dysfunction and mortality.

TTE was performed on the first day of admission. However, the circumstances differed as the same patient had already received fluids, a vasopressor infusion, or mechanical ventilation. All of which may have affected the RV function. We think that serial TTE would yield stronger evidence.

Conclusion

RV dysfunction was found in nearly one-third of the studied septic patients and is associated with over fourfold higher 28-day mortality.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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